# Report No. 177



# COLD - INDUCED VASODILATATION

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Research and Development Branch
Military Planning Division
Office of The Quartermaster General
September 1951

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Department of the Army
OFFICE OF THE QUARTERMASTER GENERAL
Military Planning Division
Research and Development Branch

Environmental Protection Section
Report No. 177

# EFFECT OF BODY THERMAL STATE ON COLD-INDUCED CYCLIC VASODILATATION IN THE FINGER

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7-64-12-001 September 1951

EFFECT OF BODY THERMAL STATE ON COLD-INDUCED CYCLIC VASODILATATION IN THE FINGER


## Abstract

a. Purpose. To determine the effect of the thermal state of the body as a whole on the cooled extremity manifesting cyclic vasodilatation. To obtain information on the basic mechanisms operating in this vascular phenomenon.

b. Summary. Four unclothed, reclining men were exposed to a warm and various cold ambient conditions for three to five hours, while one or both hands were cooled. Skin temperature and blood flow changes in the fingers were recorded simultaneously. It was found that when adequate cold was applied locally to the hand initial reduction in blood flow and objective cooling occurred, but after a short period there followed an abrupt return of blood flow concurrent with objective rewarming. Subsequent spontaneous, alternate cooling and rewarming continued in cyclic fashion. This periodically recurring vasodilatation in the cooled fingers was demonstrated not only when the body as a whole was warm, but also when it was well chilled, although the amount of local cooling necessary to evoke the response was less the warmer the body.

c. Conclusions. The finding that cold-induced digital vasodilatation occurs not only when the body is warm but also when the body is cold constitutes an hitherto unrecognized exception to the concept that the blood flow in the extremity depends on the "need" of the body as a whole to conserve or dissipate heat rather than on local thermal stimulation. The results reported here are compatible with the view that this vascular phenomenon is due to the activation of a local sensory axon reflex. The manner in which cold excites this reflex and the mechanisms whereby the thermal state of the body modifies the reaction are unknown, but an hypothesis implicating changes in skin thermal gradients is suggested.

d. Recommendations. It is believed essential that the Quartermaster Corps be cognizant of the above described "spontaneous" fluctuations in blood flow which occur in the cooled hand. This could assume importance in the testing and evaluation of measures designed to protect or warm the cooled extremity when the tests are based on subjective "tolerance times",

or arbitrary skin temperature values. Although the principle that the blood flow to the extremity is largely determined by the heat balance of the body as a whole is by no means refuted, it now requires qualification because of the demonstration that a local thermal stimulus -- adequate cold -- will induce vasodilatation in the generally vasoconstricted individual. On the basis of this report it is recommended that further investigation proceed along the following lines: (1) Determination of the extent and evaluation of this phenomenon in the foot, especially as it pertains to protection against cold injury; (2) Study of the relationship of individual variations in this vascular effect to "cold tolerance"; (3) Observation and evaluation of the reaction under field conditions; (4) Correlation of the blood flow and skin temperature changes with the attending variations in sensation as they concern the problem of dexterity in the cooled hand; (5) Continuation of research into the mechanisms of the phenomenon utilizing: (a) subcutaneous and intravascular temperature measurements to determine tissue thermal gradients, and (b) the technique of iontophoresis of various pharmacologic agents to reveal the possible action of chemical factors initiating this vasodilatation.

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## Foreword

The phenomenon of cyclic cold-induced vasodilatation studied in this report has been described previously, but its importance for the prevention of cold injury has been considered slight because the early findings were interpreted as indicating that the reaction would not develop in the presence of general body cooling. This opinion must undergo revision, since Captain Blaisdell's studies show that the "Lewis waves" do develop in chilled subjects, although the chilling does affect the skin temperature at which they appear. By many repetitions of the experiments, the effect of biological variation on interpretation has been minimized and quantitative relationships established between the "general body heat state" and the local temperatures associated with the onset of vasodilatation.

Among the immediate outgrowths of these findings must be a re-evaluation of the use of specified toe temperatures or of the toe temperature at fixed time intervals as the basis for interpreting clothing test results. Renewed impetus is given to studies of the local mechanisms operating in control of blood flow and the prevention of cold injury of the skin. The concept that the "state of central body heat determines the blood flow to the extremities" has become entrenched and at times extended too freely in evaluating cold protection and auxiliary heat requirements. This report, by establishing quantitative relationships between local and systemic factors, helps set limits to the valid but overextended concept of the dominance of central factors in peripheral vasomotor control.

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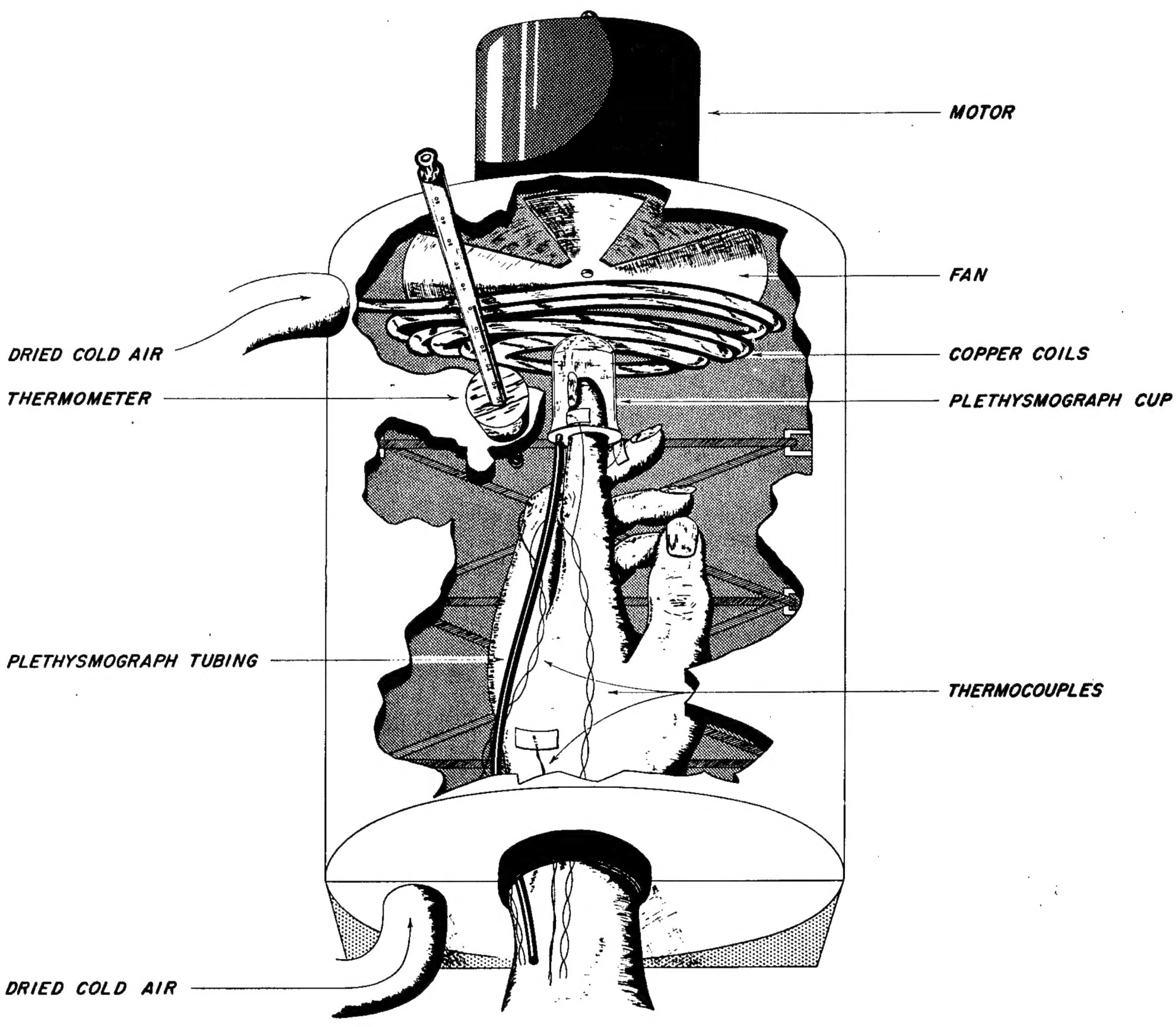
# HAND - COOLING APPARATUS




TOP VIEW

EFFECT OF BODY THERMAL STATE ON COLD-INDUCED CYCLIC VASODILATATION IN THE FINGER

1. Introduction

a. Blood flow in the cooled extremity is generally considered to be largely contingent upon thermal factors operating directly and indirectly.[1,2,3] Directly, cold induces local constriction of the superficial vessels, even in the denervated limb.[10,23,24] Indirectly, thermal stimuli over the entire body surface affect the circulation in the cooled extremity in two ways: Firstly, warm and cold influences impinging on cutaneous neuroreceptors initiate afferent impulses which reach the hypothalamic thermoregulatory center. The center responds, initiating efferent impulses transmitted by the sympathetic neural system which effect modifications in peripheral vascular tone. This reflex activity is manifested generally throughout the body, but is only transitory.[25] Secondly, thermal influences over the body surface also induce fine alterations in the temperature of the circulating blood. These modifications in blood temperature presumably act on the highly vascular hypothalamic center which promotes a sustained adjustment in peripheral blood flow mediated through the sympathetic vasomotor nerves.[9,12,25]

b. Considerable evidence has accumulated to indicate that the indirect predominate over the direct local thermal influences. Thus, the extremity may remain warm with fairly high blood flow as long as the body is adequately warm. Recent quantitative studies have shown that this state will continue as long as general net body heat loss does not exceed approximately 15 percent.[26] Furthermore, indirect digital vasodilatation may be produced, as demonstrated by marked rises in skin temperature and blood flow, in spite of local cooling temperatures as low as -5°C.[2] This evidence has engendered the conclusion that blood flow to the hands and feet depends primarily on the thermoregulatory "needs" of the body as a whole and to a considerably lesser extent on local thermal stimuli. In fact, so well entrenched in current thinking is this concept that the Quartermaster Corps utilizes it as a cardinal principle governing clothing of the soldier under various thermal stress situations.

c. In 1929 Lewis[19] reported what appeared to be a specific local vasodilating effect of cold on the digits. When a finger was placed in an ice-water bath, the skin temperature fell abruptly to low levels, then rose 3° to 10 C.°, and continued to fall and rise in cyclic fashion about every 10 to 30 minutes. He attributed occasional failures of this phenomenon to general cooling of the body by the low environmental temperatures on those experimental days. More recently there has been frequent reiteration that this vascular reaction is not in evidence when the body as a whole is in negative heat balance, when there is a "need" for body conservation of heat, and therefore the reaction has little local protective value.[2,3,8] As Day[8] writes, "It is as though the body risks peripheral frostbite in order to preserve the heat of vital central organs."

d. Suggestive evidence to the contrary is supplied by Spealman[28] who reported greater blood flow in hands immersed in water at 2°C. than in hands at 15°C., although his lightly clad subjects were in cool ambient air at 15°C. Miller[22] has demonstrated pronounced phasic skin temperature fluctuations in the extremities of heavily clothed subjects exposed to -40°C. for three to eight hours daily. Kramer and Schulze[17] and Greenfield et al[14] have also described cold-induced digital vasodilatation, although admittedly slight, in a few instances when the subjects were considered generally cool.

e. The present experiments were designed to resolve the conflicting reports on the influence of the thermal state of the body on circulation in the cooled extremity, expecially in regard to the phenomenon of cyclic vasodilatation in the digit.

## 2. Materials and Methods

### a. General design of experiments.

(1) Control of the thermal state of the body as a whole was effected by modifying the room air temperature to which the quiet, unclothed, recumbent subject was exposed. Twenty-eight degrees centigrade was selected as the "adequately warm" ambient condition, as other investigators[11,15] had shown that this temperature was within the narrow range at which the neutral body thermal state was effectively maintained. Three cold ambient conditions were employed: (a) 25°C., which was "slightly cool"; (b) 15°C., a "moderately cold" environment at which most of the general bodily physiologic responses to cold, such as shivering and marked peripheral vasoconstriction, were known to occur; and (c) 12°C., at which the subject after a two- to three-hour exposure was considered well "chilled". At room temperatures of 25°C. hand responses were observed during the process of body cooling, (i.e., during abrupt change from a warm to a cool ambient condition); while at the room temperature of 15°C., studies were conducted during the body cooling period but also after two to three hours when temperature measurements became relatively stable at the new cold condition; and at 12°C. hand measurements were made after two to three hours when the subject was considered very cold or well "chilled".

(2) Local cooling of the hand was induced by exposure of the extremity to dry cold air in an insulated box. In most of the previously cited studies, local cooling was effected by immersion of the digit or hand in cold water. It was realized that there was a considerable difference in thermal conductivity of air and water. Nevertheless, air as a medium of cooling was felt to have these advantages: it simulated field conditions; and it allowed more convenient employment of pneumoplethysmographic and skin thermocouple measurements. The box temperatures used varied in 5°C. steps from 0° to 15°C. Simultaneous finger skin temperature and pulse volume measurements were recorded.

(3) In later experiments a second hand-cooling box was used so that it was possible to cool the two hands at different rates. A study of the relationship of blood flow fluctuations occurring in opposite hands was conducted in two experiments. One hand was inserted in the hand-cooling box, and about 30 minutes later the other hand was inserted.

(4) Because Miller[22] had suggested that cyclic vasodilatation of the extremities might be a local adaptation effect that did not appear until three or four days of successive general cold exposures, the experiments were so arranged that two men were subjected to the "adequately warm" room (28°C.) initially, while one man was started at exposures to the cold ambient condition (15°C.).

b. <u>Time and place.</u>

A total of 60 experiments were performed for periods of three to five hours in the mid-morning during the winter and early spring months, February through May 1951, at the Quartermaster Climatic Research Laboratory in Lawrence, Massachusetts.

c. <u>Subjects.</u>

Four healthy enlisted men, ages 20 to 26 years, who exhibited no evidence of peripheral vascular abnormalities were used as test subjects. The past history of subjective "cold tolerance" among the men ranged from "fair" to "excellent". The subjects refrained from tobacco smoking twelve hours, from alcohol consumption 24 hours, and no food was allowed for at least two hours before the experiments were begun. The subjects were nude, except for loose-fitting cotton shorts, and rested motionless, save for instances of involuntary shivering, in the supine recumbent position throughout the test periods which lasted three to five hours.

d. <u>Controlled-temperature chamber.</u>

A constant temperature room was used for all of the studies. The following room air temperatures, controlled to ±1.0 C.°, were employed: (1) 28°C., (wet bulb, 20°C.); (2) 25°C., (wet bulb, 20°C.); (3) 15°C., (wet bulb, 12°C.); and (4) 12°C., (wet bulb, 10°C.). The room remained quiet except for the continuous "hum" of the ventilating fan and motor. Care was exercised to eliminate all exciting stimuli during the experiments.

e. <u>Hand-cooling apparatus.</u> (Illustration facing page 1.)

The hand was cooled by inserting it in a cylindrical, fiberglas-covered, wood box placed at approximate heart level. Dehumidified air, cooled by previous passage through copper coils in an insulated tank containing "dry ice", was passed into the box under pressure of about 5 lbs. per square inch.

Fairly uniform but turbulent air flow, one to two mph, within the cooling device was assured by the construction of baffles and a small motor-driven fan at the opposite end of the box. Air temperatures of 0°C., 5°C., 10°C., and 15°C.: (indicated by an alcohol thermometer placed about 3 cm. from the hand surface) were used and easily maintained within ±1.0 C.° by adjusting the ingress of cooled air. The hand, resting on a rope latticework in the box, was inserted to the level of the styloid processes of the wrist where a wool cuff fitted snugly but did not constrict blood flow. The neutral position of the hands and fingers, as illustrated, provided comfort and maximal exposure of the hand skin surface without undue vascular compression.

f. Stretcher and canopy.

The subject in all experiments lay on a standard-type, wheeled, hospital stretcher. In certain instances, when it was desired to study the responses while the body was in the process of cooling, a stretcher-canopy combination was used. In these experiments, the subject assumed the recumbent position on the stretcher outside the cold chamber. After being fitted with thermocouples and plethysmograph cups, a specially constructed, lightweight, fiberglas-covered canopy was placed over the stretcher completely inclosing but not disturbing the subject. The canopy air temperature (not controlled) was found to be between 27° and 31°C. on different days. Baseline measurements (temperature and plethysmograph recordings) were made when the subject reached "thermal equilibrium", i.e., after about 30 to 60 minutes, as indicated by fairly constant rectal and surface temperature readings including a toe temperature above 32°C. When there was difficulty attaining an adequate degree of body warmth, the subject was covered with a double layer of standard Army wool blanket. Upon reaching "thermal equilibrium" the subject was wheeled into the cold room where the canopy was removed effecting an abrupt exposure of the entire body to the cool environment.

g. Temperature measurements.

(1) Finger temperatures.

Surface temperatures were measured with No. 30 wire copper-constantan thermocouples fixed to the skin with a single layer of adhesive tape and connected by way of a cable and selector switch to a Leeds & Northrup manual potentiometer. The reference junction was maintained at 0°C. in an ice-water bath. The thermocouples in all experiments were attached to the second and third digits (index and middle fingers) of each hand. The finger thermocouple was fixed to the dorsum of the terminal phalanx just proximal to the nail, a site selected because of its relatively uniform keratin layer. Maintained attachment was further assured by special "molding" of the thermojunction so as to fit the convexity of the finger. The temperatures of other fingers were also measured in special instances. Temperature readings were recorded every two and one-half minutes.

(2) Rectal temperatures.

Using a modification of the rigid type Mead-Bommarito thermocouple rectal catheter[21], the temperature of the anterior rectal wall 7 cm. from the anal sphincter was read on the above potentiometer every five or ten minutes in all experiments.

(3) Total body temperature measurements.

At cold ambient conditions of 15° and 12°C., a rough index of the magnitude of the total body heat loss after two to three hours of cold exposure was determined by computing "heat debt" (body heat production, i.e., $O_2$ consumption, however, was not measured) using the formula proposed by Burton[6].

$$\text{Heat debt (kilogram-calories)} = \frac{T_{\Delta} \times 0.83 \times W}{SA}$$

where:

$T_{\Delta}$ = the change in mean body temperature

0.83 = specific heat of human tissue

$W$ = weight of the subject in kilograms

$SA$ = surface area of the subject in square meters

The mean body temperature ($T_B$) was determined by "weighting" rectal and average skin temperatures as given by the formula:

$$T_B = 0.67\ T_R + 0.33\ T_S$$

where:

$T_R$ = rectal temperature

$T_S$ = mean skin temperature

The mean skin temperature was obtained by measuring the temperature of the following skin regions with a 10-point thermocouple harness and proportioning each according to the corresponding percent of surface area:

| Body Region | Percent of Surface Area |
|---|---|
| Forehead | 10.0 |
| Chest | 12.5 |
| Back | 12.5 |
| Upper Arm | 7.0 |
| Forearm | 7.0 |
| Hand | 6.0 |
| Inner Thigh | 12.5 |
| Outer Thigh | 12.5 |
| Foreleg | 15.0 |
| Foot | 5.0 |

h. Finger blood flow.

(1) The Burch-Winsor portable model of the digital pneumoplethysmograph designed by Turner[29] was utilized to indicate changes in finger circulation. Five cubic centimeters of the terminal phalanx of the finger were inclosed without compression in the cellulose-acetate cup and sealed with a synthetic adhesive, Pliobond. The plethysmograph sensitivity was adjusted so that a 10 mm. deviation on the tracing was equivalent to a 10 cu. mm. volume change of the 5 cc. segment of finger. The completed plethysmogram presented a continuous recording of pulse volume, showing alterations in vascular tone of the tissue part, and, as Burton[7] has demonstrated a useful though only roughly quantitative index of actual blood flow.

(2) A plethysmograph cup was fitted to the index finger of each hand. Early experiments indicated that there was a slight though definite insulating effect due to the cup. The finger skin temperature at which cold-induced vasodilatation appeared and the height of the vasodilatation waves averaged 2 C.° and 3 C.° higher, respectively, than those of an uncupped finger. The responses in the uncupped adjacent (third) finger of each hand therefore served as the control for this artefact.

(3) Pulse volume values in the finger of a thermally neutral subject (at a room temperature of 28°C.) were found to vary with frequent spontaneous fluctuations from 3 to 15 cu. mm. per 5 cc. of finger. Nevertheless, changes due to cold-induced vasodilatation waves were easily distinguishable by their pronounced and sustained magnitude. In the cooled vasoconstricted finger, values below 1 cu. mm. per 5 cc. of finger were difficult to measure with accuracy and were therefore plotted as "less than one" and considered "minimal".

3. Results

a. General description of cold-induced cyclic vasodilatation in the finger.

In general, when the hand was placed in the hand-cooling box, the cooled fingers manifested the following changes in the plethysmogram and skin temperature recordings as graphically presented in Figure 1.

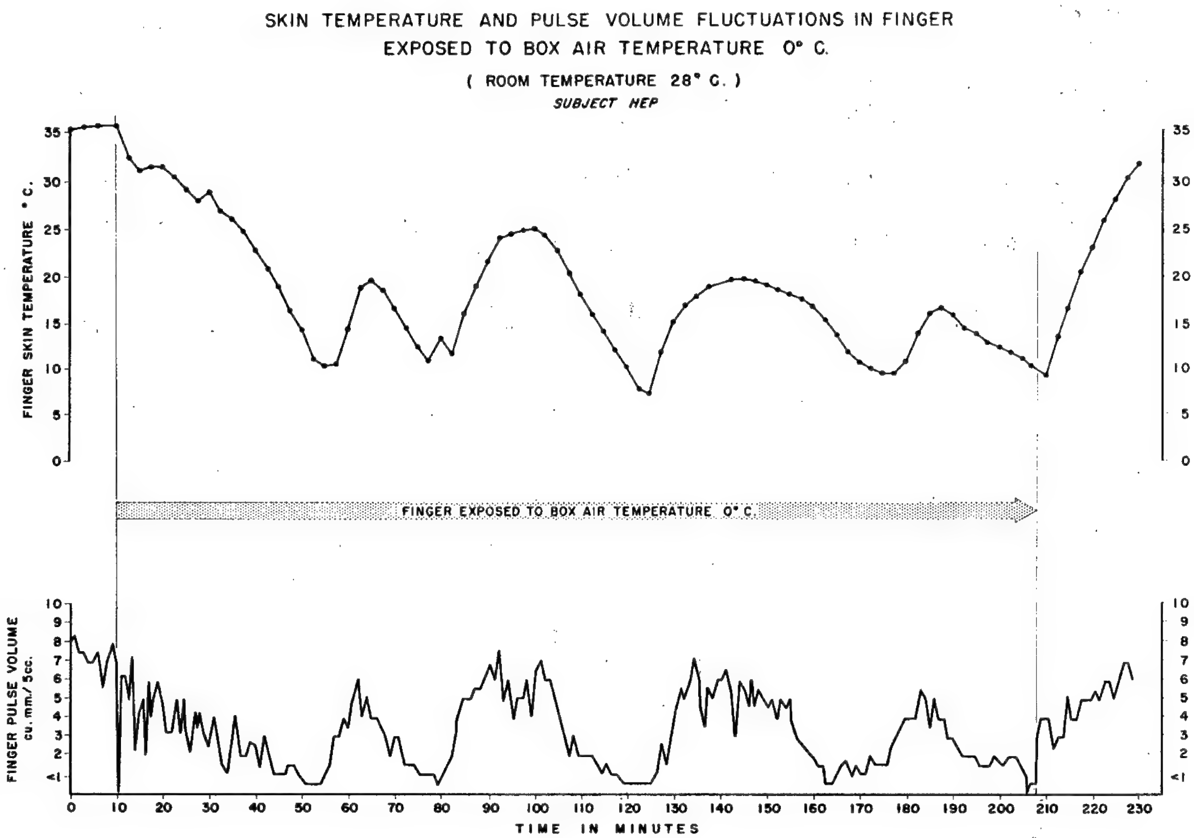



FIGURE 1

Initially there was a transitory fall in pulse volume followed by a more gradual reduction (with wide and frequent fluctuations) until minimal values (less than 1 cu. mm.) were reached; and a decline in skin temperature, lagging behind the vascular adjustments, to a nadir approaching the box air temperature. After a 5- to 20-minute period of maximal vasoconstriction and objective cooling, the following sequence (reverse of the above) spontaneously ensued: There was an abrupt re-opening of constricted vessels as revealed by a sharp increase in pulse volume and a rise in skin temperature. This new state of vasodilatation persisted for 5 to 80 minutes, after which gradual cooling again set in. Subsequent periodic increases in blood flow and skin temperature recurred

every 10 to 110 minutes. Local cold exposures of the hand were continued for as long as five hours during which as many as seven successive cold-induced vasodilatation waves were observed, yet there was no tendency for these oscillations to become attenuated or less frequent.

b. <u>Cold-induced vasodilatation in the fingers of the warm and cool individual.</u>

Finger skin temperature and pulse volume changes in a hand subjected to local air at a temperature of 0°C. were observed in three subjects exposed to room temperatures of 28°C. ("warm"), 25°C. ("slightly cool"), and 15°C. ("moderately cold"). In 15 experiments on Subject H.E.P., 10 experiments on Subject R.D.L. and 4 experiments on Subject J.A.L., vasodilatation waves in the cooled fingers were exhibited in every instance at all three room conditions -- not only when the body was comfortably warm (room temperature 28°C.) but even when the body was cooling at ambient temperatures of 25°C. and 15°C.

c. <u>Cold-induced vasodilatation in the fingers of the "chilled" individual.</u>

The possibility that this digital vascular reaction might not occur in an individual who was further cooled to the extent that he could be considered thoroughly "chilled" was explored in four subjects. They remained at room temperatures of 15°C. or 12°C. for two to three hours prior to insertion of a hand in the cold box which was at 0°C. Body surface and rectal temperature measurements were recorded at varying intervals during the cooling period for calculation of total body heat debt. Table I presents the heat debt data obtained in seven experiments at the time of insertion of the hand and in some instances before and after this event. The control values recorded when the body was "warm" (room temperature 28°C.) are also given for comparison. Figure 2 shows the time course of events during one of the tests. After about one hour generalized shivering was pronounced. In one and one-half hours extremity skin temperatures had fallen to near that of the room where they "leveled off"; finger pulse volume fluctuations were no longer detectable and mean body surface temperatures had dropped to about 25°C. A paradoxical rise in rectal temperature of the cooled person was noted in two tests. In two experiments an attempt was made to enhance body cooling by the oral administration of 500 cc. of ice water. In one instance the rectal temperature promptly fell 0.2°C. ; in the other it remained unchanged for 20 minutes and then slowly dropped 0.2°C. . In spite of the tendency of the rectal temperature to remain fairly stable, calculated average body temperatures at the end of the body-cooling period (110 to 183 minutes) had fallen 1.5 to 3.6°C. and the individual was "chilled" to an extent indicated by heat debt values of 52.8 to 113.7 kg-cal/m.$^2$. Even under these conditions of body chilling with marked generalized peripheral vasoconstriction, further cooling of the hand by exposing it to air at 0°C. was followed by finger vasodilatation in all seven experiments on four subjects.

TABLE I: TOTAL BODY HEAT DEBT DURING TWO-TO THREE-HOUR COLD EXPOSURES AT 15° OR 12°C. (Four Subjects)

| Subject | Room Temp. | Time After Onset of Body Cold Exposure | Mean Skin Temp. $T_S$ | Rectal Temp. $T_R$ | Mean Whole Body Temp. $T_B$ | Change in Mean Whole Body Temp. $T_\Delta$ | Calculated Heat Debt |
|---|---|---|---|---|---|---|---|
| | °C. | min. | °C. | °C. | °C. | °C. | kg-cal/m.² |
| | 28 | 110 | 32.2 | 37.1 | 35.5 | 0.0 | 00.0 |
| | 28 | 180 | 32.2 | 37.1 | 35.5 | 0.0 | 00.0 |
| | | 84 | 26.1 | 37.0 | 33.4 | -2.1 | 63.8 |
| H.E.P. | 12 | 115 | 25.7 | 37.1(+) | 33.3 | -2.2 | 66.8 |
| | | 195 | 25.1 | 37.0 | 33.0 | -2.5 | 76.0 |
| | | 75 | 26.9 | 36.8 | 33.5 | -2.0 | 60.8 |
| | | 105 | 26.1 | 36.8 | 33.2 | -2.3 | 69.9 |
| | 12 | 145 | 25.7 | 36.9(+) | 33.2 | -2.3 | 69.9 |
| | | 210 | 25.8 | 36.8 | 33.1 | -2.4 | 72.9 |
| | 28 | 115 | 34.2 | 36.9 | 35.9 | 0.0 | 00.0 |
| | | 121 | 27.6 | 37.0 | 33.9 | -2.0 | 69.2 |
| R.D.L. | 15 | 183 | 27.4 | 37.0 | 33.8 | -2.1 | 72.7 |
| | | 250 | 26.4 | 36.8 | 33.3 | -2.6 | 90.0 |
| | 12 | 140 | 24.8 | 36.6 | 32.7 | -3.2 | 110.8 |
| | | 190 | 24.9 | 36.4 | 32.6 | -3.3 | 114.2 |
| | 28 | 110 | 32.9 | 37.0 | 35.4 | 0.0 | 00.0 |
| | | 97 | 26.5 | 37.6 | 33.9 | -1.5 | 49.5 |
| G.T.G. | 15 | 110 | 26.5 | 37.4 | 33.8 | -1.6 | 52.8 |
| | | 197 | 26.2 | 37.4 | 33.7 | -1.7 | 56.1 |
| | 12 | 110 | 25.2 | 38.0 | 33.7 | -1.7 | 56.1 |
| | | 165 | 25.2 | 37.8 | 33.6 | -1.8 | 59.4 |
| J.A.L. | 28 | 110 | 34.1 | 37.1 | 36.4 | 0.0 | 00.0 |
| | 12 | 110 | 24.9 | 36.8 | 32.8 | -3.6 | 113.7 |

Underscored values indicate conditions existing just prior to insertion of hand in cold air at 0°C.

(+) Paradoxical rise in rectal temperature during cold exposure.

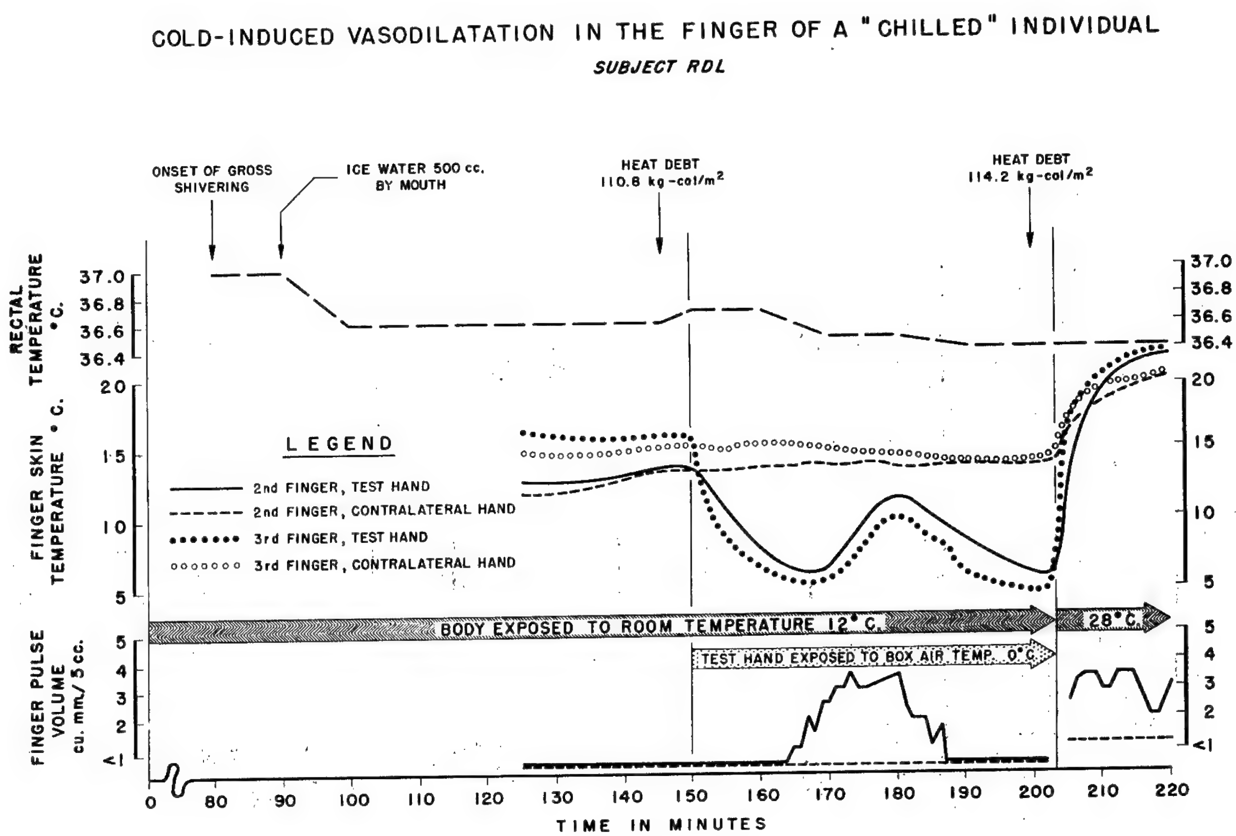



FIGURE 2

d. <u>Critical local (hand) and general (body) thermal conditions for cold-induced vasodilatation.</u>

(1) The results of the initial experiments suggested that although cold-induced vasodilatation was not prevented by general body cooling, the finger skin temperature at which the reaction appeared was influenced by the thermal state of the body -- the warmer (or less cool) the body the higher the local skin temperature at which the vasodilatation wave would "take off"; and likewise, the cooler the body the lower this local critical skin temperature. Statistical comparison of group means of the finger skin temperature at which vasodilatation waves began (hereafter referred to as "nadir temperature") strengthened this impression as shown in Table II. The mean nadir finger skin temperature when the body was warm (room temperature 28°C.) was 7.8°C. (±3.5) while at the cooler ambient conditions the average finger temperatures were significantly lower, 3.7°C. (±1.9) to 5.7°C. (±2.1) when vasodilatation occurred. The values observed when the body was "moderately cold" were not significantly lower than those obtained when the body was "slightly cool". Similarly the differences when the body was "moderately cold" and "chilled" were not significant, but when comparison was made when the body was "slightly cool" as opposed to "chilled", significantly different values were apparent. These data show that the surface temperature at which a vasodilatation wave appeared tended to be lower the cooler the body, although at the coolest ambient conditions the distinction was not as clearly evident.

TABLE II: FINGER SKIN TEMPERATURE AT WHICH COLD-INDUCED VASODILATATION APPEARED ("NADIR TEMPERATURE") AT VARIOUS BODY THERMAL STATES (Three Subjects)

| Room Temperature | Hand-Cooling Box Temperature | Number of Nadir Temperature Points | Range of Nadir Temperature Points | Mean Nadir Temperature (±1 S.D.) |
|---|---|---|---|---|
| °C. | °C. | | °C. | °C. |
| 28 "Adequately Warm" | 0 | 106 | 1.0 to 15.4 | 7.8 (±3.5) |
| 25 "Slightly Cool" | 0 | 125 | 0.0 to 13.0 | 5.7 (±2.1) |
| 15 "Moderately Cold" | 0 | 29 | 1.0 to 9.3 | 5.1 (±2.3) |
| 15 "Chilled" | 0 | 10 | 1.0 to 6.8 | 3.7 (±1.9) |
| 12 | 0 | 9 | 2.3 to 6.7 | 3.8 (±2.0) |

Each mean nadir temperature value was found to be significantly different from all others at the 5% probability level with the following exceptions: "Slightly Cool" and "Moderately Cold"; "Moderately Cold" and "Chilled".

(2) It was thought that if a certain local temperature were reached before vasodilatation occurred, it should be possible to demonstrate that cooling one hand to this critical thermal state would induce local vasodilatation while less, "inadequate" cooling of the contralateral hand would not evoke this response. Further, these differential hand-cooling conditions should vary depending on the body thermal state. The following experiment based on this rationale was performed. Three men were subjected to various local (hand) and general (body) thermal conditions as shown in Table III, while finger temperature and pulse volume data were collected. At room temperatures of 15°C. and 25°C. the hand (No. 1) exposed to the cooler air manifested definite phasic vasodilatation while the contralateral hand (No. 2) remained persistently vasoconstricted. An example is shown in Figure 3. At room temperature of 28°C. blood flow fluctuations appeared in the hand (No. 1) at air temperatures of 5°C., while oscillations occurred irregularly or equivocally in the opposite hand (No. 2) cooled at 15°C. It seemed apparent that a "moderately cold" room condition (15°C.) the critical local temperature or "adequate" cold stimulus was in the neighborhood of 5°C., while air

TABLE III: CRITICAL LOCAL (HAND) COLD EXPOSURE TEMPERATURE AT WHICH COLD-INDUCED VASODILATATION IN THE FINGER OCCURRED AT VARIOUS GENERAL (BODY) THERMAL STATES (Three Subjects)

| Room Temperature | Subject | Hand No. 1 | | Hand No. 2 | |
|---|---|---|---|---|---|
| | | Temperature to which Exposed | Finger Vaso-dilatation? | Temperature to which Exposed | Finger Vaso-dilatation? |
| °C. | | °C. | | °C. | |
| 15 | H.E.P. | 5 | Yes | 10 | No |
| "Moderately | R.D.L. | 5 | Yes | 10 | No |
| Cold" | J.A.L. | 5 | Yes | 10 | No |
| 25 | H.E.P. | 10 | Yes | 15 | No |
| "Slightly | R.D.L. | 10 | Yes | 15 | No |
| Cool" | J.A.L. | 10 | Yes | 15 | No |
| 28 | H.E.P. | 5 | Yes | 15 | No |
| "Adequately | R.D.L. | 5 | Yes | 15 | Equivocal |
| Warm" | J.A.L. | 5 | Yes | 15 | Yes |

at 10°C. was "inadequate"; when the body was "slightly cool" (room temperature 25°C.), a local air temperature of 10°C. was clearly sufficient to evoke cyclic vasodilatation while 15°C. was not; and when the body was "warm" (ambient temperature 28°C.) slight fluctuations occurred occasionally at a local cooling temperature as high as 15°C.

e. Effect of body thermal state on height and duration of cold-induced vasodilatation waves.

Table IV presents the data on the height and duration of the finger skin temperature oscillations measured on four subjects at various body thermal conditions when the hand was in a cold box at 0°C. The largest waves (temperature increments as great as 31°C.) occurred when the body was warm, while at the cooler room conditions the rise in finger temperature, although conspicuous, was never greater than 16.5°C. . However, the variability at each condition as can be seen in the table was wide and analysis failed to reveal any correlation of height of the vasodilatation waves with the various room temperatures. In like manner, the duration of the vasodilatation waves tended to be longer when the room was less cool, but no consistent or systematic relationship with the room temperature was evident.

COLD-INDUCED VASODILATATION IN "ADEQUATELY" COOLED FINGER BUT NOT IN "INADEQUATELY" COOLED FINGER
( ROOM TEMPERATURE 25° C. )
SUBJECT RDL

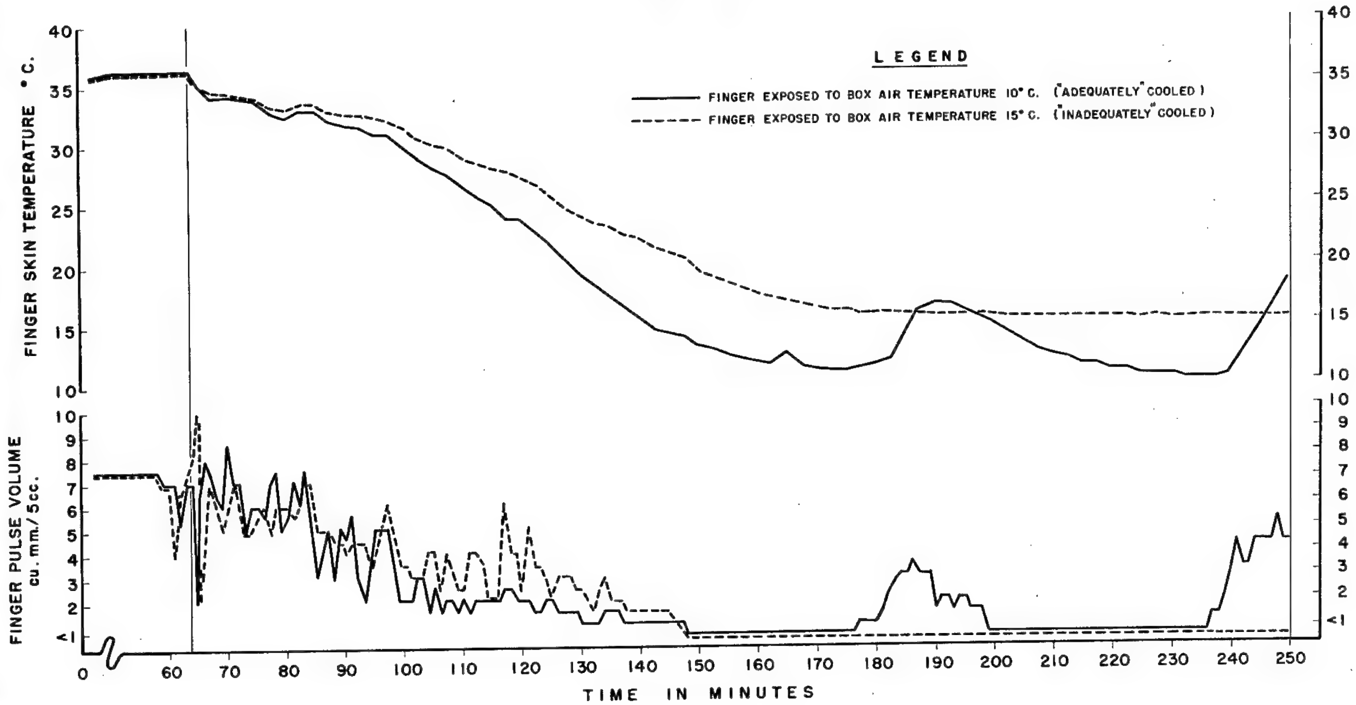



FIGURE 3

f. <u>Effect of the thermal state of the contralateral hand on cold-induced vasodilatation waves.</u>

In Table V are the tabulated results on the finger skin temperature at which vasodilatation waves appeared, (nadir temperature), their height and duration when the contralateral hand was exposed to various local air temperatures from 0° to 28°C., when the room temperature was 28°C. The mean values at each condition are seen to follow no consistent pattern. Nadir temperatures were not lower when the contralateral hand was cooler. This is in contrast to the above reported effect of the body thermal state on these values.

TABLE IV: HEIGHT AND DURATION OF COLD-INDUCED VASODILATATION WAVES IN THE FINGER AT VARIOUS BODY THERMAL STATUS
(Four Subjects)

| Thermal Conditions | | Height of Vasodilatation Waves | | | Duration of Vasodilatation Waves | | |
|---|---|---|---|---|---|---|---|
| Room Temp. | Hand-Cooling Box Temp. | No. of Waves Obs. | Range | Mean (±1 S.D.) | No. of Waves Obs. | Range | Mean (±1 S.D.) |
| °C. | °C. | | °C. | °C. | | min. | min. |
| 28 "Adequately Warm" | 0 | 88 | 3.5 to 31.0 | 9.7 (±5.5) | 86 | 15 to 70 | 34 (±16) |
| 25 "Slightly Cool" | 0 | 85 | 3.5 to 15.0 | 7.3 (±3.3) | 79 | 10 to 100 | 48 (±22) |
| 15 "Moderately Cold" | 0 | 19 | 3.5 to 15.5 | 6.6 (±3.7) | 19 | 15 to 40 | 26 (±8) |
| 15 "Chilled" | 0 | 9 | 5.0 to 16.5 | 10.1 (±4.0) | 8 | 26 to 38 | 31 (±4) |
| 12 | 0 | 9 | 4.0 to 15.0 | 9.1 (±3.8) | 7 | 16 to 27 | 23 (±4) |

g. Central and local control of cold-induced vasodilatation.

(1) Although the above data indicate that vasodilatation appeared in the fingers only when cooled "adequately" and therefore this intermittent phenomenon was probably due to locally acting mechanisms, occasionally apparent synchronism of vasodilatation waves in adjacent fingers suggested that some central factor might be controlling the reaction.

(2) Information on this point was obtained by simultaneous temperature measurements of all fingers of the cooled hand, and by observation of skin temperature and pulse volume changes in the index finger of each hand, when one hand was exposed to cold (0°C.) 30 minutes later than the other. The wholly discordant skin temperature oscillations of the five fingers of the cooled hand are shown in Figure 4. Likewise the previously cooled hand had no effect on the frequency of fluctuations in temperature or pulse volume occurring in the hand inserted after a delay of 30 minutes (Figure 5). The waves started out of phase and demonstrated no tendency to "catch up" or "get in step".

TABLE V: EFFECT OF THERMAL STATE OF CONTRALATERAL HAND ON COLD-INDUCED VASODILATATION WAVES IN FINGERS OF TEST HAND

(Four Subjects)

| Thermal Conditions | | | Finger Skin Temperature at Which Vasodilatation Waves Appeared | | | Height of Vasodilatation Waves | | | Duration of Vasodilatation Waves | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Room Temperature | Box Air Temperature | | | | | | | | | | |
| | Test Hand | Contralateral Hand | Number of Waves Observed | Range | Mean | Number of Waves Observed | Range | Mean | Number of Waves Observed | Range | Mean |
| °C. | °C. | °C. | | °C. | °C. | | °C. | °C. | | min. | min. |
| 28 | 0 | 28 | 43 | 1.0 to 13.0 | 7.0 | 36 | 3.5 to 31.0 | 11.4 | 33 | 15 to 80 | 39 |
| 28 | 0 | 10 | 15 | 2.0 to 13.5 | 7.6 | 11 | 3.0 to 23.0 | 10.9 | 11 | 18 to 50 | 28 |
| 28 | 0 | 0 | 34 | 2.0 to 15.5 | 7.9 | 33 | 3.0 to 18.0 | 8.5 | 29 | 15 to 60 | 26 |
| 28 | 10 | 28 | 6 | 11.5 to 15.0 | 13.6 | 5 | 2.5 to 20.0 | 10.0 | 5 | 10 to 25 | 19 |
| 28 | 10 | 0 | 5 | 11.0 to 12.5 | 12.0 | 5 | 3.0 to 16.5 | 9.4 | 5 | 30 to 40 | 37 |

h. Cold-induced vasodilatation as a possible adaptation effect.

The possibility that cyclic vasodilatation in the extremity might be a local "acclimatization" effect or that modifications in this phenomenon might be apparent after successive exposures was also studied. Two men were exposed to the "warm" room condition (28°C.) first and then to successively cooler room temperatures, while one man was started at the cold room condition (15°C.) and then successively exposed to less cool room temperatures. Finger vasodilatation resulting from "adequate" local cold stimulation appeared in the first experiment in all three subjects. Statistical observation of the test data at a given thermal condition did not indicate any modifications in the finger skin temperature at which vasodilatation waves appeared, nor alterations in their height and duration throughout the course of the experiments. These data did not lend themselves to convenient presentation in tabular or graphic form and accordingly are purposely omitted.

i. Inadvertent frostbite.

Although the tests were not directed toward the study of cold injury, it is deemed worthy to report observations of three separate instances of frostbite which inadvertently occurred. The room temperature on these occasions was 28°C. Shortly after insertion of the hand in the cold box, the box air temperature fell below -2°C. Finger skin temperatures fell rapidly. There was attending extreme cold and pain sensation which after a short time was replaced by sudden numbness. At this point skin temperature readings were discovered to be less than 0°C. and the hand was quickly withdrawn from the box. The finger was seen to exhibit the typical white wheal of frostbite. These cases of acute cold injury occurred during the initial local cooling phase. In no instance were cold-induced vasodilatation waves observed prior to the discovery of frostbite.

4. Discussion

a. The results reported here demonstrate that local cold of sufficient intensity will uniformly induce finger cyclic vasodilatation when the body is warm and thus confirm previous studies.[3,14,17,18,19] However, the finding that even when the body is cooling or thoroughly "chilled", further cooling of the hand will evoke local vasodilatation in the face of generalized maximal vasoconstriction is contrary to generally accepted views.[2,3,8] The basis for these former opinions has long rested on evidence usually obtained during the course of experiments investigating other phenomena. In such instances tests were terminated when surface temperatures dropped to what were considered dangerously low levels or when cold and/or pain became "intolerable". Under these circumstances the vasodilatation reaction was in all probability overlooked. The report of Grant et al[13] that a similar vascular phenomenon occurring in the rabbit's ear fails to appear when the rectal temperature falls below 38°C. due to general body cooling has also contributed to this widely held view.

COLD-INDUCED VASODILATATION WAVES OCCURRING INDEPENDENTLY IN FINGERS OF HAND EXPOSED TO BOX AIR TEMPERATURE 0° C.

( ROOM TEMPERATURE 25° C. )

*SUBJECT HEP*

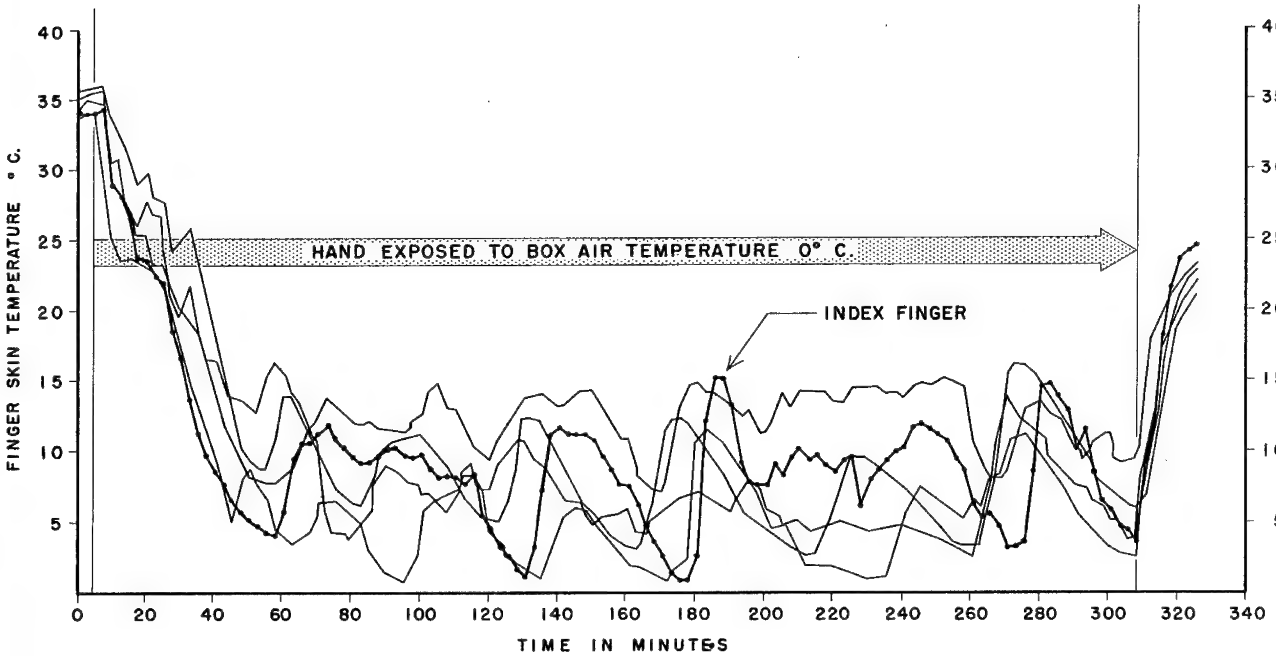



FIGURE 4

COLD-INDUCED VASODILATATION WAVES OCCURRING INDEPENDENTLY IN INDEX FINGER OF EACH HAND

( RIGHT HAND EXPOSED TO BOX AIR TEMPERATURE 0°C. THIRTY MINUTES AFTER EXPOSURE OF LEFT HAND )

( ROOM TEMPERATURE 28° C. )

*SUBJECT JAL*

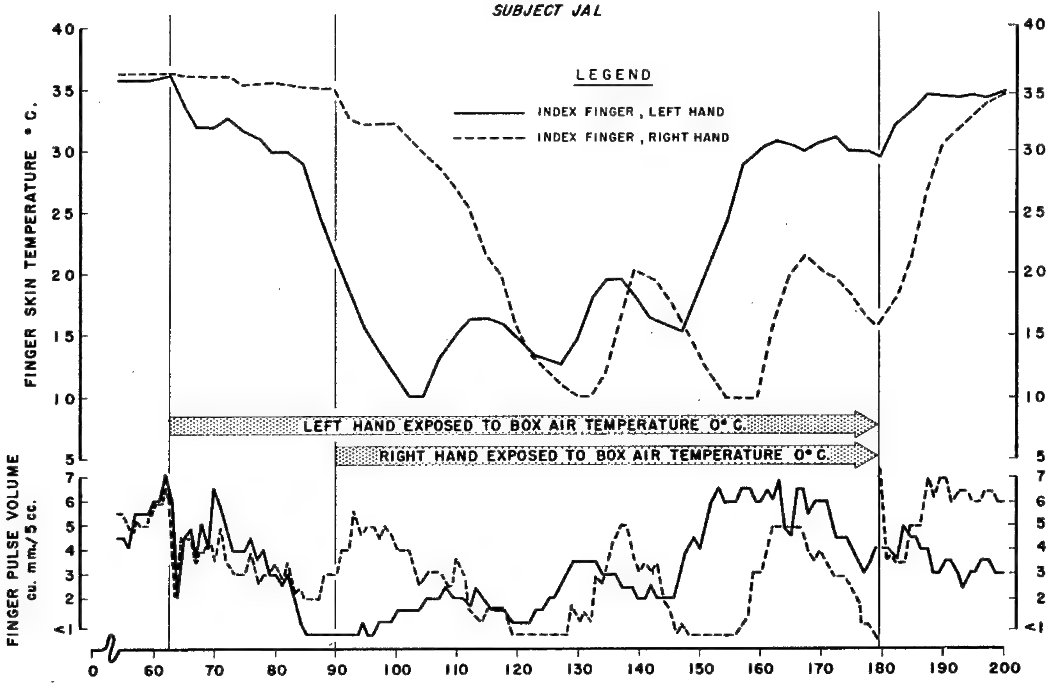



FIGURE 5

b. Objection may be made to the present study that inasmuch as rectal temperatures never fell below 36.4°C., the subjects were not really cold. However, within the range of physiologic responses to cold the temperature of the rectum is notoriously poor as an index of body cooling, for it remains quite stable above 36°C. or may even rise paradoxically, seemingly as a result of the heat-producing efficiency of shivering.[26] In this report the nude men were subjectively very uncomfortable to the point of generalized involuntary rigors, and objectively their digital skin temperatures approximated the room temperature while finger plethysmography indicated maximal peripheral vasoconstriction. It was not feasible to induce hypothermia to the extent revealed by rectal temperatures of 35°C. or less, a condition at which shivering ceases and hyperreflexia, dysarthria and delayed cerebration appear.[16]

c. It is however evident that within the range of general bodily physiologic adjustments to cold, this peripheral vasodilatation effect is still elicitable. This finding constitutes an important exception to the oft-cited maxim that blood flow to the extremities depends on the "need" of the body to conserve or dissipate heat rather than on local thermal influences.[2,25] The local vasodilatation occurs in spite of the bodily "need" to remain generally vasoconstricted.

d. Spealman's report[27] that blood flow in the hand is greater at local exposure temperatures of 2°C. than at 15°C. has led to the inference that the relationship is constant. The results reported here based on continuous pulse volume and surface temperature recordings indicate that heightened blood flow at "adequately" cold conditions is an intermittent, cyclic phenomenon. The difference in blood flow between an "adequately" cooled and "inadequately" cooled extremity depends on the point during a vasodilatation wave at which the comparison is made. At the crest of a wave the difference will be great, but at a nadir there may be no difference, both values being minimal.

e. The suggestion of Miller[22] that cold-induced vasodilatation was an example of local "acclimatization" inasmuch as it did not appear until after three or four days of successive exposures is not supported here. In this study vasodilatation waves were demonstrated on the first exposures to cold without a tendency to modification in subsequent exposures. The initial absence of the phasic temperature fluctuations in Miller's experiments may be explained by the probable apprehension and anxiety of the subjects during early exposures at -40°C., an effect which Bader and Mead[2] have well shown, may have, in addition to cold, a strong and sustained vasoconstricting action.

f. The demonstration that vasodilatation waves appeared independently in separate fingers and without correlation with fluctuations in contralateral cooled fingers suggests the operation of a specific local mechanism induced by adequate chilling. This evidence does not support

Kunkle's view[18] that these oscillations may be regarded as gross exaggerations of "spontaneous" rhythmic fluctuations in blood flow occurring synchronously in the digits of subjects resting at neutral thermal conditions.[5,7]

g. Lewis[19] in human subjects and Wybauw[30] working with the cat have demonstrated that the vascular reaction to cold occurs in the limb after section of sympathetic and somatic sensory nerves but not after degeneration of sensory axones. Thus the participation of a local sensory axon reflex appears to be essential. The manner in which cold activates this reflex is not clear. Lewis contended that the adequate stimulus is a critical concentration of "H-substance" released by cells injured by cold, but this concept is difficult to reconcile with the discovery of these waves at local cooling temperatures as high as 15°C. at which cell damage seems unlikely. The possibility that vasoconstriction, which may be maximal at this temperature, may induce ischemic cellular injury and thus release of a metabolite stimulating this reflex is considered. However, this also appears doubtful for the present studies indicate that a finger may be maximally vasoconstricted, yet if not "adequately" cooled will not demonstrate cyclic vasodilatation.

h. In spite of the foregoing evidence seeming to establish the vasodilatation response to cold as a purely local phenomenon, the additional finding that the local surface temperature at which vasodilatation occurs is related to the degree of body warmth, introduces a factor of central influence and suggests a possible clue as to the mechanism by which the local axon reflex is awakened. The upper limit of cooling that induces a response is about 15°C., but at this local condition the body must be warm; if the body also be cooled at an ambient temperature of 15°C. finger vasodilatation does not appear. Further local cooling of the hand at about 5°C. is then necessary to induce vasodilatation. Accordingly, if an extremity be cooled sufficiently, vasodilatation waves appear, but the degree of local cooling necessary to evoke the reaction is less the warmer the body. The fact that cooling of the contralateral hand does not influence the reaction in the test hand, while cooling of the entire body does, indicates that a fairly large part of the body surface exerts this central effect. It thus appears that a certain critical temperature difference must obtain between the general body thermal state and the locally cooled hand.

i. How does this temperature difference between the body and the extremity stimulate a local axon reflex? Since central neural connections are not essential for this reaction, the alternative mediating pathway is the circulating blood. A critical temperature difference between the entering warm blood and the surface of the cooled finger setting up a sufficiently steep spatial thermal gradient at the site of axon reflex receptors might conceivably constitute the adequate stimulus initiating local reflex vasodilatation. Lewis[20] reported that

cold-induced finger vasodilatation was prevented when the proximal arm was cooled in water at 15°C. A possible interpretation would be that the arterial blood in the forearm was precooled by the water bath to the extent that when the blood reached the digit, which was also cool, an adequate thermal gradient did not exist and therefore the reflex was not excited. On this basis the following hypothesis is proffered: As the finger is cooled a thermal gradient, between the entering relatively warm arterial blood and cool skin, is established at the depth of neuroreceptors. When this gradient achieves sufficient magnitude it triggers a local axon reflex which induces arterial dilatation and rewarming of the finger. With the resulting influx of warmer blood, the cooling effect on the skin surface is overcome and the tissue tends to become uniformly warm. The thermal gradient between the superficial and deep tissues is reduced and the reflex vasodilatation is no longer maintained. The finger cools and vasoconstricts until an adequate gradient is again restored. If the body be warm, the temperature of the blood entering the finger is relatively high and mild surface cooling suffices to establish an adequate gradient. The local reflex rewarming therefore begins at a relatively high skin temperature. Likewise cooling of the body as a whole lowers the temperature of the blood entering the finger, and therefore only by reducing further the surface temperature by a greater amount of local cooling is an adequate gradient set up. At these conditions activation of the axon reflex and resulting vasodilatation appear at a relatively lower skin temperature.

j. An evaluation of this local vasodilating effect of cold is now partially permissible. Fingers chilled, maximally vasoconstricted and near freezing have been shown to rewarm spontaneously. The local "need" to rewarm by vasodilatation predominates over the general body "need" to conserve heat by maintenance of peripheral vasoconstriction. It seems in order, therefore, to ascribe local protective value against severe finger cooling to this phenomenon. However, it has not been demonstrated here that this reaction protects against cold injury. In the three instances when the hand box air temperatures dropped below -2°C., frostbite appeared during initial cooling without preceding vasodilatation waves. There was no evidence that the pathologic change resulted from "exhaustion" of cyclic vasodilatation. Kramer and Schulze[17] have reported finger vasodilatation at local cooling air temperatures as low as -18°C. with finger surface temperatures falling to nadirs of -2°C. at the onset of vasodilatation. No cases of frostbite were mentioned. Confirmation of their work and demonstration that frostbite would result when vasodilatation waves no longer appeared would constitute evidence establishing this reaction as a local protective mechanism against cold injury.

k. Cognizance of this phenomenon makes the use of "tolerance times" based on arbitrary skin temperature values or on subjective sensations hazardous, e.g., in differentiating the efficacy of protective gear for hands and feet. It is likely on such tests that skin temperatures previously considered "illogical", "obviously incorrect" or attributed to

spurious local exercise, because they indicated a warm rather than a cool extremity, were in reality due to this spontaneous rewarming effect.

5. Summary

a. A study was undertaken to determine the effect of the thermal state of the body as a whole on the cooled extremity, especially one manifesting cyclic vasodilatation, and by correlation of finger skin temperature and blood flow seek understanding of the mechanisms involved in the phenomenon.

b. Four unclothed, reclining men were exposed to a warm and various cold ambient conditions for three to five hours while one or both hands were differentially cooled.

c. It was found that:

(1) Provided adequate cold was applied to the hand, spontaneous, intermittent vasodilatation occurred in the fingers.

(2) These phasic changes in blood flow were readily demonstrated not only when the body as a whole was warm but when it was actively cooling and even when it was considered well "chilled".

(3) The degree of local cold necessary to evoke the cyclic response was less the warmer the body.

(4) The thermal state of the contralateral hand, however, had no observable effect on the oscillations in the fingers of the test hand.

(5) The cyclic changes in blood flow occurred independently in separate fingers and without correlation with fluctuations in contralateral cooled fingers.

(6) Cold-induced vasodilatation in the fingers was demonstrated in the first experiment in all subjects regardless of the body thermal state. The vasodilatation waves at a given thermal condition showed no tendency to modification throughout the course of the experiments.

(7) Three instances of inadvertent frostbite occurred during the initial cooling phase of the hand. No vasodilatation waves were observed prior to the discovery of frostbite.

6. Conclusions

a. Contrary to generally accepted views, cold-induced vasodilatation in the finger occurs not only when the body as a whole is warm but also when the body is cooling or is thoroughly "chilled". This constitutes an hitherto unrecognized exception to the concept that the blood flow to

the extremities depends on the thermoregulatory "needs" of the body as a whole rather than on local thermal stimulation.

b. The reaction is a specific local vasodilatation due to cold, modified but not wholly determined by the body thermal state.

c. Cold-induced vasodilatation in the finger is not a local adaptation or "acclimatization" phenomenon.

d. The evidence reported is compatible with the view that this specific effect of cold results from the activation of a local sensory axon reflex. The manner in which cold awakens this reflex and the mechanism whereby the body thermal state influences the reaction are unknown but an hypothesis implicating local changes in tissue thermal gradients is suggested.

e. The reaction is interpreted as being a local protective mechanism against severe cooling of the finger.

7. Acknowledgements

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d. The statistical analyses were performed by Miss J. A. Klimas, and the figures were drafted by Sfc. A. M. Hilgendorf, to whom the author is also indebted.

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1 Ft. Totten Medical Center, Ft. Totten, Long Island, N.Y. (Attn: Chief Eng. Dev. Div., Army-Navy Med. Procurement Off.)

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